

In the United States Court of Federal Claims
OFFICE OF SPECIAL MASTERS
No. 16-922V
Filed: March 28, 2024

*
L.R., by and through his parent and *
natural guardian, MICHELE BAXTER, *
*
Petitioner, *
*
v. *
*
SECRETARY OF HEALTH AND *
HUMAN SERVICES, *
*
Respondent *

Maximillian Muller, Muller Brazil, LLP, Dresher, PA, for Petitioner
Madelyn Weeks, U.S. Department of Justice, Washington, DC, for Respondent

DECISION ON ENTITLEMENT¹

Oler, Special Master:

On August 2, 2016, Michele Baxter (“Petitioner”) filed a petition for compensation under the National Vaccine Injury Compensation Program, 42 U.S.C. § 300aa-10, *et seq.*² (the “Vaccine Act” or “Program”) on behalf of her minor grandson L.R. The petition alleges that L.R. developed anti-N-methyl-D-aspartate receptor (“anti-NMDAR”) encephalitis as a result of the measles-

¹ Because this Decision contains a reasoned explanation for the action in this case, it must be made publicly accessible and will be posted on the United States Court of Federal Claims' website, and/or at <https://www.govinfo.gov/app/collection/uscourts/national/cofc>, in accordance with the E-Government Act of 2002. 44 U.S.C. § 3501 note (2018) (Federal Management and Promotion of Electronic Government Services). This means the Decision will be available to anyone with access to the internet. In accordance with Vaccine Rule 18(b), Petitioner has 14 days to identify and move to redact medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. If, upon review, I agree that the identified material fits within this definition, I will redact such material from public access.

² National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755. Hereinafter, for ease of citation, all “§” references to the Vaccine Act will be to the pertinent subparagraph of 42 U.S.C. § 300aa (2012).

mumps-rubella (“MMR”), tetanus-diphtheria-acellular pertussis (“Tdap”), hepatitis B, inactivated poliovirus (“IPV”), and varicella vaccines he received on August 4, 2014.

Upon review of the evidence in this case, I find that Petitioner has failed to preponderantly demonstrate that the vaccination was the cause of L.R.’s condition. The petition is accordingly dismissed.

I. Procedural History

Petitioner filed her petition on August 2, 2016, followed by L.R.’s medical records. Pet. at 1; Ex. 2, 3, 4. Respondent filed his Rule 4(c) Report on January 17, 2017 recommending that entitlement be denied. Resp’t’s Rep. at 1; ECF No. 16.

The parties then submitted expert reports from their respective experts in neurology. Petitioner filed expert reports from Dr. Lawrence Steinman. Ex. 11, 12, 13. Respondent filed expert reports from Dr. Eric Lancaster. Ex. A, C, D, E, F.

I conducted an entitlement hearing on January 12-13, 2022. The parties filed post-hearing briefs followed by a joint status report indicating that the record is complete. ECF No. 80. This matter is now ripe for adjudication.

II. Anti-NMDAR Encephalitis

Anti-NMDAR encephalitis is a disease defined by autoantibodies to the NMDA receptor. First Lancaster Rep. at 15. These autoantibodies are directly pathogenic and disrupt the receptor’s functioning. *Id.* Anti-NMDAR encephalitis causes “changes of mood, behavior, and personality, resembling acute psychosis.” Florance et al., *Anti-N-Methyl-D-Aspartate Receptor (NMDAR) Encephalitis in Children and Adolescents*, 66 ANN NEUROL. 1, 1-15 (2009) (filed as Ex. A, Tab 3). Most patients develop a progressive illness that often begins with “psychosis, memory deficits, seizures [and] language disintegration” and develops into “a state of unresponsiveness with catatonic features...” Dalmau et al., *Clinical experience and laboratory investigations in patients with anti-NMDAR encephalitis*, 10 LANCET NEUROL. 1, 63-74 (2011) (filed as Ex. A, Tab 2) (“Dalmau”). More than 75% of patients experience “substantial recovery” after immune therapy that takes place in the reverse order of symptom onset. Dalmau at 1.

There is no dispute that anti-NMDAR encephalitis is L.R.’s correct diagnosis. Tr. at 74 (Dr. Steinman). Tr. at 146 (Dr. Lancaster).

III. Medical Records

The parties do not dispute the accuracy of Petitioner’s medical history as documented in the medical records. Joint Pre-Hearing Submission at 2; ECF No. 53.

L.R. developed a strep throat infection at the end of July, 2014 that was treated with antibiotics. *See, e.g.* Ex. 3 at 41; Ex. 3 at 2262; Ex. 3 at 2391; Ex. 3 at 2246.

L.R. was seen by his primary care doctor on August 4, 2014, for his four-year-old check-up. There were no behavioral problems noted, but he did have eating difficulties. His blood pressure was high (121/77). The developmental screen noted he only counted to one and did not name any colors, but gross motor development was normal. The exam showed allergic shiners (dark circles under the eyes), but was otherwise normal. MMR, varicella, DTaP, and hepatitis B, and IPV vaccines were given. Ex. 1 at 1-2.

On August 8, 2014, L.R. was seen in the Lincoln County Emergency Room for altered mental status. Ex. 2 at 1-3. The record is difficult to read, but seems to indicate that L.R. was not acting like himself, was staring at the ceiling, and was not responsive, so his mother called EMS. *Id.* at 1.

L.R. was seen in the Vanderbilt ER on August 9, 2014, at 3 a.m., with the complaint that he was not acting like himself, was paranoid, seeing people, had green diarrhea, and was waking up every hour kicking and screaming since receiving immunizations on Monday. Ex. 3 at 41-42. The attending from the ER, Dr. Suzanne Bryce, noted that for one week L.R. had altered behavior during the night, awakening and inconsolable, and was yelling about demons without mouths. He was completely normal during the day, with no abnormal behavior, and no hallucinations. She noted a history of strep throat two weeks prior with complete recovery and four year old vaccinations on Monday. L.R. had been staying up until midnight lately to wait for his mother's boyfriend to come home from work and he did have a history of frequent nightmares. He underwent a CT of the head and a urine drug screen at an outside hospital, both of which were negative. His exam and gait were normal. The assessment was that in the absence of fever and in the absence of symptoms in the daytime, encephalitis, meningitis, post-infectious disorders, or structural causes were unlikely. He was diagnosed with night terrors. Ex. 3 at 41-42. L.R. was discharged on August 13, 2014.

L.R. returned to the Lincoln County ER on August 14, 2014, with a chief complaint of twitching that had been occurring over the past two days. Ex. 2 at 4. The record is difficult to read. It appears to state the following under "past [history]":

Grandmother stated that Pt had vaccine shots and 4 days later started having seizure like activity was crying at night [illegible] bilateral leg pain. Was admitted and discharged. Pt now continues with twitching and jerking movement of bilateral legs and unable to walk.

Id. The clinical impression is listed as "tics." *Id.* at 5. L.R. was transferred to the Vanderbilt ER.

L.R. arrived at the Vanderbilt ER on August 15, 2014, at 2 a.m. for evaluation of tremors, twitching, hand flapping, and emotional outbursts. His vital signs showed blood pressure of 134/87. Ex. 3 at 2242-45. The history indicated that he had eight days of involuntary movements and hallucinations. After being diagnosed with night terrors, L.R. developed clumsiness, symptoms of obsessive-compulsive disorder ("OCD"), and ataxia and was admitted at an outside hospital and given antibiotics and treated for agitation. The history also included a strep throat infection three weeks prior and then vaccinations two weeks prior. Multiple family members had

strep throat around the same time. CBC, electrolytes, CRP, urinalysis, and urine toxicology screen were negative/normal. *Id.* at 2262-64, 2271.

On August 15, 2014, neurology consulted on L.R.'s case. L.R. had a one week history of crying and thrashing during sleep and then clumsiness, unsteadiness, and emotional lability in the setting of recent vaccinations and a strep throat infection. He had been admitted at an outside hospital where he required benzodiazepines to calm him down and he received antibiotics for an elevated WBC count. The parent noted the last few days he had been unsteady on his feet and had "OCD tendencies." Ex. 3 at 2246-47. There had been no seizure activity, no recent fever, no known ingestions. The exam noted that he was unable to keep his arms or legs still, he had normal strength, normal reflexes, normal sensation, and no ataxia. He could take five to six steps without support. L.R. was diagnosed with chorea; given the recent strep infection and psychiatric symptoms, the physicians believed his presentation was consistent with Sydenham's chorea. He was to receive high dose penicillin. Ex. 3 at 2246-49.

L.R. was admitted to Vanderbilt Hospital from August 15 to October 23, 2014. He was started on treatment with gabapentin and Depakote³ for his chorea, emotional lability/rage, and unsteadiness. The neurologist stated even if antibody tests were negative for strep, they would still treat with prophylactic penicillin until age 21 for rheumatic fever. Ex. 3 at 2274-76. By August 18, 2014, when the antibodies for strep were negative, the treating physicians started to investigate causes of chorea in general. L.R. also was started on Klonopin⁴ for his symptoms of emotional lability, but the medication was later changed to Depakote. *Id.* at 2294, 2335. A lumbar puncture performed on August 20, 2014, showed a white blood cell count of 12 with 4 neutrophils, 87 lymphocytes, with glucose 59 and protein 2. The CSF red blood cell count was 761. *Id.* at 2341. The CSF NMDA receptor antibody test was positive at 1:40. *Id.* at 2302.

On August 18, 2014, L.R.'s ASO and anti-DNase-B⁵ tests returned as negative. Ex. 3 at 2292.

³ Depakote: "trademark for a preparation of divalproex sodium." DORLAND'S MEDICAL DICTIONARY ONLINE (HEREINAFTER "DORLAND'S"), <https://www.dorlandsonline.com/dorland/definition?id=13254> (last accessed March 13, 2024); Divalproex sodium: "a coordination compound of valproate sodium and valproic acid in a 1:1 molar relationship, used in the treatment of manic episodes associated with bipolar disorder and epileptic seizures, particularly absence seizures, and the prophylaxis of migraine; administered orally." DORLAND'S, <https://www.dorlandsonline.com/dorland/definition?id=14613> (last accessed March 13, 2024).

⁴ Klonopin: "trademark for a preparation of clonazepam." DORLAND'S, <https://www.dorlandsonline.com/dorland/definition?id=27123> (last accessed March 13, 2024); Clonazepam: "a benzodiazepine used as an anticonvulsant in the treatment of Lennox-Gastaut syndrome and of atonic and myoclonic seizures and as an antipanic agent in the treatment of panic disorders; administered orally." DORLAND'S, <https://www.dorlandsonline.com/dorland/definition?id=10139> (last accessed March 13, 2024).

⁵ ASO or antistreptolysin O titer. *Mosby's Manual of Diagnostic and Laboratory Tests 6th Ed.* at 420. Anti-DNase-B or antideoxyribonuclease-B Titer. *Id.* at 421. These tests are used to identify antecedent infection by group A streptococcal bacteria. *Id.*

On August 21, 2014, L.R. started complaining of neuropathic pain in his feet; an EMG/NCS performed the next day was normal. Ex. 3 at 2355-56. He had an EEG on August 24, 2014, which was normal, and a video EEG that was non-epileptic. *Id.* at 2351, 2388. During this time, L.R.'s speech was largely unintelligible, and he was having poor sleep with concerns for delirium or psychosis. *Id.* at 2374.

On August 25, 2014, the anti-NMDA receptor serum antibody test was positive. Ex. 3 at 2405. Rheumatology consulted on August 25, 2014, and felt that his presentation was concerning for anti-NMDA encephalitis and planned to start the process of ruling out a paraneoplastic cause. *Id.* at 2421-23. On August 26, L.R. had an MRI of the chest, abdomen, and pelvis which ruled out a tumor. *Id.* at 2471. An infectious disease consult on August 27 recommended ruling out TB, viral/arthroviral encephalitis, bartonella, histoplasma, and EBV infections prior to starting steroids and IVIG, but felt that the presentation was most consistent with anti-NMDAR encephalitis. *Id.* at 2483-88. A repeat lumbar puncture on August 29 again showed a positive anti-NMDA receptor titer of 1:40. *Id.* at 2532. On August 30, L.R. started treatment with a pulse dose IV steroids for three days, then a standard dose of steroids and IVIG every two weeks. *Id.* at 2597.

On September 4, 2014, L.R. was scheduled to start plasmapheresis for five days and then rituximab every two weeks for two total doses in addition to the daily steroids. Ex. 3 at 2695. From September 12 to October 23, 2014, L.R. recovered on the pediatric floor prior to transfer to rehabilitation ("rehab"), and his caregivers noted slow improvements. *Id.* at 2836. L.R. started physical, occupational and speech therapies ("PT/OT/ST") and continued to make slow improvements, he was fed through a nasojugal ("NJ") tube, and psychiatry followed his catatonia closely. Ex. 3 at 2892, 2975. Because of poor oral motor control and not tolerating swallowing foods, a G-tube was placed on October 15, 2014. *Id.* at 3442.

L.R. transferred to inpatient rehab at Cincinnati Children's on October 23, 2014, with one further treatment of IVIG and a plan to complete weaning from steroids. Ex. 3 at 3584-88. After a month, L.R. was not progressing and he was transferred to inpatient care for another five plasma exchanges. He then he returned to therapy/inpatient rehab for another six weeks. Ex. 3 at 3655; Ex. 4 at 13. His hospital course was also complicated by an umbilical abscess and an influenza infection. In rehab, he improved to tolerate a regular diet, walked, played, and spoke in two to three word sentences. Ex. 5 at 44. He was discharged home on January 2, 2015. Ex. 4 at 14.

L.R. saw his rheumatologist in the clinic on January 7, 2015. He still had emotional lability and aggressiveness. He was continuing to attend PT, OT, and ST. His exam showed slightly slurred speech with a loud volume. Ex. 5 at 44-46.

L.R. was seen by his primary care doctor, Dr. Cline, on January 19, 2015. Dr. Cline noted the diagnosis of anti-NMDA encephalitis and that L.R. was being followed by rheumatology, psychiatry, and neurology at Vanderbilt and Cincinnati, and was getting PT, OT and ST. He was taking lorazepam, melatonin, clonidine, gabapentin, and Risperdal. He was receiving some feedings through a G-tube. A physical exam noted that his speech was slurred but was otherwise normal. Ex. 1 at 12-13.

L.R. returned to the rheumatology clinic on February 11, 2015. He had possible worsening of his language skills. A repeat lumbar puncture on February 27, 2015, showed an anti-NMDAR antibody titer of 1:10. Ex. 3 at 3607. The level was reassuring to rheumatology and they did not plan any additional therapy. *Id.* at 3659. He returned to the rheumatology clinic on March 11, 2015, at which time his behavior and nightmares continued to be an issue. *Id.* at 3661.

L.R. was seen by pediatric neurology on April 14, 2015. He had a slow improvement back to baseline physically and had been discharged from PT. He still had speech articulation issues and fine motor skill deficits. Emotionally, he was easily frustrated, violent on occasion, cried easily, and was hyperactive. The school had planned to delay his enrollment in kindergarten for a year given his behavioral problems. His exam was normal except for mild dysarthria. Neurology recommended starting psychiatric medications to improve his daily functioning. Ex. 3 at 3655-57.

At follow-up visit with rheumatology on April 23, 2015, L.R. was still having tantrums at home, but his behavior was good at school. He was on clonidine and gabapentin. They noted that his autoimmune process was not active. He had full return of B cells and was not immunosuppressed. Ex. 5 at 57.

An undated note from Clifford Seyler, M.D., of Royal Pediatrics, stated that L.R. underwent chemotherapy after his diagnosis and had immune suppression and could not receive any further vaccines. He was excused from all classes during outbreaks of flu and strep. Ex. 3 at 3668.

L.R. had his first visit with Dr. Seyler on July 13, 2015. He noted attention problems, disruptive sleep, frustration, and incontinence. He noted no problems with previous immunizations but that Cincinnati advised that he should receive additional vaccines because of his compromised immune system. Ex. 7 at 13-16.

L.R. returned to the rheumatology clinic on July 31, 2015. L.R. was still having periods where he would get upset and hyperventilate. He was participating in behavioral therapy. His exam was normal. The assessment indicated that there was no active disease process and that he was not immunosuppressed and could receive normal vaccines. He was starting kindergarten with plans for an individual education plan (“IEP”). Ex. 5 at 62-63. A follow-up note on January 15, 2016, indicates that his G-tube was removed in October and he was not taking any medications. *Id.* at 64.

On October 20, 2015, L.R. received a flu shot. The note indicates that his rheumatologists agreed that he should receive it. Ex. 7 at 8-9.

A note from the behavioral therapist on December 22, 2015, noted that L.R. was going to school with an IEP in place which included speech services. His main issue was frustration and melt-downs at home with his grandmother. Ex. 6 at 18.

IV. Petitioner’s Affidavit and Testimony

A. Petitioner’s Affidavit

Petitioner signed her affidavit on December 20, 2021. Ex. 19. In it, she averred that she is L.R.'s biological grandmother and his natural guardian. *Id.* at 1. Prior to vaccination, L.R. had strep throat and recovered with antibiotics. *Id.*

After L.R. received his vaccinations on August 4, 2014, he complained of leg pain and was overly emotional, crying while watching cartoons. Ex. 19 at 1. He also began having night terrors. *Id.* L.R. was taken to the Lincoln Medical Center emergency room on August 8, 2014 for hallucinations and difficulty talking; he was discharged with a prescription for valium. *Id.* L.R. was taken to Vanderbilt (Children's Hospital) the following day due to continued nightmares and screaming. *Id.* He was diagnosed with night terrors and was discharged from Vanderbilt. *Id.*

On August 14, 2014, L.R.'s mother called an ambulance because L.R. was having hallucinations and had lost control of his bodily functions. Ex. 19 at 2. He was initially taken to the Lincoln ER and then was transferred to Vanderbilt. *Id.* During his stay at Vanderbilt, L.R. was unable to walk, eat, or drink. *Id.* L.R. was hospitalized for two months. *Id.* L.R. was moved between the pediatric department and the ICU multiple times due to his fluctuating condition. *Id.* L.R. was seen by a rheumatologist who suggested a spinal tap to diagnose encephalitis and was started on plasmapheresis, IVIG, and a chemotherapy drug. *Id.* L.R. was transported to Cincinnati Children's Hospital for treatment as he was not improving at Vanderbilt. *Id.* at 3. L.R. underwent additional rounds of plasmapheresis and IVIG while hospitalized in Cincinnati. *Id.*

Eventually, L.R. "snapped out of it and woke up." Ex. 19 at 3. L.R. was able to walk and swallow again and improved rapidly. *Id.* After a few weeks of therapy, L.R. was released from Cincinnati Children's Hospital. *Id.* L.R. still had behavioral issues after his hospitalization and was diagnosed with ODD, ADHD, borderline autism, severe learning disability, and more recently Tourette's syndrome. *Id.*

B. Petitioner's Testimony

Petitioner testified at the entitlement hearing. Petitioner's daughter is L.R.'s mother. *Id.* L.R. lived with her at all times relevant to this proceeding (at time of vaccinations and currently). Tr. at 5-6.

Petitioner stated that L.R. was a well-mannered child prior to vaccination, whose health was relatively normal for a young child. Tr. at 7. Prior to the vaccination at issue, L.R. had strep throat and completed a course of amoxicillin. *Id.* at 7-8. On the day of vaccination, Petitioner had taken L.R. and her daughter to Dr. Cline, L.R.'s pediatrician, for his four-year old wellness check. *Id.* at 9. Petitioner had an errand to run so she returned after 15 minutes and waited in the car for L.R. and her daughter to return but noticed that they were taking longer than expected. *Id.* at 10. At some point, Petitioner went into the office to check on L.R., when she heard him screaming from the hallway. *Id.* Petitioner recalled L.R.'s screaming was from receiving the vaccinations in his legs and he subsequently complained of leg pain and had a fever over the following days. *Id.* at 10-11.

L.R. complained of leg pain for about four days after the vaccination. Tr. at 58, 59. Petitioner would carry him when he complained of pain and put cold rags on his legs because they were red and swollen. *Id.* at 58. Petitioner was unsure when the leg pain ended in conjunction with L.R.'s walking difficulties, which occurred about a week after vaccination. *Id.* at 60.

On August 8, 2014, Petitioner received a phone call from L.R.'s mother at around 11:00pm; L.R. was having hallucinations. Tr. at 12. Petitioner told L.R.'s mother to call 911. *Id.* at 13. Petitioner drove to L.R.'s mother's house just as L.R. was being loaded into the ambulance. *Id.* Petitioner remembered that L.R. was hallucinating, could not "hold his bowels," was "foaming at the mouth," and had an elevated blood pressure. *Id.* Some tests were run on L.R. but he was discharged with Ativan.⁶ *Id.*

The following morning at 3:00am on August 9, 2014, L.R. was taken to Vanderbilt Children's Hospital because Petitioner thought that Lincoln Medical Center had not done enough to help him. Tr. at 13-14. L.R. was discharged from Vanderbilt on the same day. *Id.* at 15. Later, on the same day, L.R. was admitted once again to Lincoln for "screaming, pushing, and hitting." *Id.* at 15. L.R. was admitted to Lincoln Medical Center for four days. *Id.* at 16.

On August 14, 2014, Petitioner and L.R. returned to the Lincoln ER. Tr. at 19. L.R.'s legs would buckle and he could not ambulate well. *Id.* at 20. Petitioner drove L.R. to Vanderbilt from the Lincoln ER rather than opting for an ambulance. *Id.*

Petitioner testified about L.R.'s two-month stay at Vanderbilt Children's Hospital. Petitioner stated that L.R. continued to decline, he had problems walking and after a few days, also lost the ability to drink liquids. Tr. at 23. L.R. was losing sensation in his feet and was also experiencing catatonia. *Id.* at 24.

On August 23, 2014, L.R.'s treating physicians began to suspect L.R. had anti-NMDA encephalitis. Tr. at 27. The physicians began treatment, which involved plasmapheresis, IVIG, rituximab, and steroids. *Id.*

On September 4, 2014, L.R.'s testing was positive for anti-NMDAR encephalitis and he was started on new medications. *Id.* at 31. L.R. was transported to Cincinnati Children's Hospital by ambulance, but did not improve much while he was hospitalized there. Tr. at 39-40.

⁶ Ativan: trademark for preparations of lorazepam. DORLAND'S, <https://www.dorlandsonline.com/dorland/definition?id=4704> (last accessed on January 23, 2024); Lorazepam: a benzodiazepine with anxiolytic and sedative effects, administered orally in the treatment of anxiety disorders and short-term relief of anxiety symptoms and as a sedative-hypnotic agent, and intravenously or intramuscularly for preanesthetic medication; used also intravenously to control status epilepticus and as an antiemetic in cancer chemotherapy. DORLAND'S, <https://www.dorlandsonline.com/dorland/definition?id=28747> (last accessed on January 23, 2024).

Petitioner stated that a doctor suggested one final treatment for L.R., Cytosan,⁷ which was a chemotherapy treatment and more potent than rituximab. Tr. at 40. Given its potency, Ms. Baxter was unsure whether to give L.R. the treatment. *Id.* at 40-41. While Ms. Baxter was deciding whether to give L.R. Cytosan, he woke up. *Id.* at 41-42.

Petitioner stated that L.R. underwent therapy and tests but had seemingly returned to normal; he could walk, run, and swallow again. Tr. at 42. After being discharged, L.R. still suffered from violent outbursts. *Id.* at 43. L.R. was also still wearing diapers and used a G-tube for feeding. *Id.*

L.R. went to kindergarten with an individualized education program (IEP). Tr. at 49. Petitioner recalled that L.R. did not enjoy school; he had panic attacks and cried frequently; he wore diaper which was embarrassing for him; L.R. also refused to get out of the car when being dropped off at school and would wrap the seat belt around his neck to keep from going to school. *Id.* at 49-50.

Petitioner stated that L.R. is very active with fishing and riding a bike; but he still has emotional outbursts though they are infrequent. Tr. at 55. L.R. was recently diagnosed with Tourette Syndrome and has facial tics. *Id.* He is taking guanfacine and sertraline. *Id.*

V. Expert Opinions and Qualifications

A. Dr. Lawrence Steinman

1. Qualifications

Dr. Steinman attended Dartmouth College and Harvard Medical School. Ex. 12.1 (hereinafter “Steinman CV”) at 1. Dr. Steinman completed his residency at Stanford and a neuroimmunology fellowship at the Weizmann Institute in Israel. *Id.* Dr. Steinman is board certified in neurology. *Id.* at 2. He has taught neurology, pediatrics, and genetics since 1980 and is currently a professor at Stanford University in the departments of neurology, pediatrics, and genetics; he is also the George A. Zimmermann Professor of Neurological Sciences at Stanford University. *Id.* at 1. Dr. Steinman has approximately 50 patents and has published approximately 600 peer-reviewed papers. *Id.* at 2-46; Tr. at 64. Dr. Steinman was elected to the National Academy of Medicine and National Academy of Science; volunteer for jobs that help U.S. Government. Tr. at 65. He chairs a research advisory committee for Gulf War illness. *Id.* at 66.

At the time his first expert report (in 2017), Dr. Steinman had seen about five patients with

⁷ Cytosan: trademark for preparations of cyclophosphamide. DORLAND’S, <https://www.dorlandsonline.com/dorland/definition?id=12503> (last accessed on January 24, 2024); cyclophosphamide: a cytotoxic alkylating agent of the nitrogen mustard group, used as an antineoplastic, often in combination with other agents, for a wide variety of conditions... also used as an immunosuppressive agent to prevent transplant rejection and in the treatment of certain diseases with abnormal immune function. Cyclophosphamide itself is pharmacologically inert; several active metabolites are produced by the microsomal enzyme systems in the liver. DORLAND’S, <https://www.dorlandsonline.com/dorland/definition?id=12166> (last accessed on January 24, 2024).

anti-NMDA encephalitis; he now sees three to four cases per year. Tr. at 67-68. He has been asked to consult on cases and estimates he sees approximately 10 cases of anti-NMDA encephalitis each year. *Id.* at 68. I recognized Dr. Steinman as an expert in neurology and immunology. *Id.* at 69.

2. Expert Opinion

Dr. Steinman testified on both days of the entitlement hearing and has submitted three expert reports in this case. Exs. 11 (hereinafter “First Steinman Rep.”), 12 (hereinafter “Second Steinman Rep.”), and 13 (hereinafter “Third Steinman Rep.”).

Dr. Steinman defined an encephalopathy as an injury to the brain, that could come from an infection, a biochemical substance, or physical head injury. Tr. at 70. Anti-NMDAR encephalitis is an immune-related neurological disease, where the body’s immune system makes an antibody that attacks the NMDA receptor in the brain. *Id.* To diagnose anti-NMDA encephalitis, tests are done to see if antibodies to the NMDA receptor exists. *Id.* at 71. Typical presentation of anti-NMDA encephalitis begins with seizure status epilepticus or behavioral changes; presentation can vary. *Id.* at 71-72.

Dr. Steinman’s theory involves molecular mimicry. Dr. Steinman posited that L.R. was injected in the thigh with the MMR and varicella vaccines which triggered the body’s immune system to produce T and B cells to fight the viruses introduced by the vaccines. Tr. at 75. Amino acid sequences from the MMR and varicella vaccines share similarities to amino acid sequences on NMDA receptors. When an immune response is generated to protect the host after vaccination, the immune system attacks both the viruses introduced by the vaccines and the NMDA receptors, which causes anti-NMDAR encephalitis. First Steinman Rep. at 5.

According to Dr. Steinman, the vaccines at issue in this case are more potent because they contain live attenuated viruses, rather than other types of vaccines that are either inactivated, mRNA, conjugate, or toxoid vaccines. Dr. Steinman emphasized his belief that anti-NMDAR encephalitis is an autoimmune response that involves T and B cells, as opposed to Dr. Lancaster who believes that it is a solely B-cell mediated process. Second Steinman Rep. at 6-7. Third Steinman Rep. at 3. More specifically, Dr. Steinman opined that T-cells initiate the B-cell response; both B and T-cells are found in the perivascular regions⁸ in patients with NMDAR encephalitis. Second Steinman Rep. at 9-10; *see also* Ex. 12.4 Camdassanche et al., *Brain immunohistopathological study in a patient with anti-NMDAR encephalitis*, EUROPEAN JOURNAL OF NEUROLOGY 2011, 18: 929–31.

Dr. Steinman further opined that T cells are “required to recognize a complex 3-dimensional structure, known as a ‘conformational epitope’,” such as the GluN1 subunit. Second Steinman Rep. at 15-16. After T cells read the linear sequences, they “proliferate” B cells to

⁸ Perivascular spaces: spaces, often only potential, that surround blood vessels for a short distance as they enter the brain; their inner wall is formed by a prolongation of a membrane like such as the arachnoid, and the outer wall by a continuation of the pia; the intervening channel communicates with the subarachnoid space. DORLAND’S, *Perivascular spaces*, <https://www.dorlandsonline.com/dorland/definition?id=107316> (last accessed on February 13, 2024).

conformational epitopes. *Id.* at 18. Dr. Steinman was hesitant to state that the N368/G369 region of the GluN1 subunit is the only conformational specificity for anti-NMDAR encephalitis; the authors of this paper seem unsure if the conformational components are at the 368-369 region. *Id.* at 17. Dr. Steinman cited to the Manca paper to support the notion that T helper cells primed with native or denatured antigen can help production of conformational antibodies. Second Steinman Rep. at 15-16; *see also* Ex. 12-9 (Manca et al., *Constraints in T-B cooperation related to epitope topology on E. coli β -galactosidase I. The fine specificity of T cells dictates the fine specificity of antibodies directed to conformation- dependent determinants*, EUROPEAN JOURNAL OF IMMUNOLOGY 15:345, 1985).

Dr. Steinman performed a BLAST search to see if there are any similarities between the vaccine components and the NMDA receptors because that would support the overarching theory that the vaccines caused L.R. to develop anti-NMDA encephalitis. Tr. at 79-85. Once overlap between the vaccine and the NMDA receptors are discovered, Dr. Steinman performs a medical literature search to see if the amino acid sequences are cited in other medical literature and if it can cause an immune response. *Id.* at 82-83.

In Dr. Steinman's first report, he was able to identify an eight amino acid sequence (QYPPTRFG) in the rubella virus that shares 7 of 8 amino acids with NMDA-R (QYPPFRFG). First Steinman Rep. at 11. This was the most significant match he found. He also found a match between the mumps virus and NMDA-R that share five of six amino acids (VRGVWG). *Id.* at 12. Dr. Steinman found other amino acid sequences which had less overlap. *See id.* at 13-17.

Dr. Steinman supports his molecular mimicry theory with the Gautam papers, which he co-authored. According to Dr. Steinman, the Gautam papers demonstrate that homology in amino acid sequences was significant enough to cause neuroinflammation or experimental autoimmune encephalomyelitis ("EAE") in mice. Ex. 11.14 (Gautam et al., *A viral peptide with limited homology to a self-peptide can induce clinical sign of experimental autoimmune encephalomyelitis*, 161 JOURNAL OF IMMUNOLOGY at 60, 60-64 (1998) (filed as Ex. 11, Tab 14) (hereinafter "Gautam 1") at 1. Researchers introduced herpesvirus saimiri ("HVS") which has "mimics" of the myelin basic protein into mice and observed neuroinflammation and demyelination. *Id.* Dr. Steinman emphasizes that these papers show that significant homology of five to six amino acid sequences can trigger or exacerbate an autoimmune response. Tr. at 85.

Dr. Steinman testified that papers cited by Dr. Lancaster show that the measles and mumps viruses can cause anti-NMDAR encephalitis. Tr. at 73-74; *see also* Armangue et al., *Pediatric anti-N-methyl-D- aspartate receptor encephalitis-clinical analysis and novel findings in a series of 20 patients*, JOURNAL OF PEDIATRICS, 2013;162(4):850-856.e2. (filed as Ex. C, Tab 3) (hereinafter "Armangue"); and Cai et al., *Anti-N-methyl-D aspartate receptor encephalitis associated with acute Toxoplasma gondii infection: a case report*, MEDICINE, 2018;97(7):e9924 (filed as Ex. C, Tab 4) (hereinafter "Cai"). Dr. Steinman stated that would indicate the vaccine that contains both viruses should also trigger the same autoimmune reaction. Tr. at 101-03. Dr. Steinman further elaborated that the Armangue and Cai papers did not mention streptococcus, which Dr. Lancaster mentioned as a potential cause of anti-NMDAR encephalitis. *Id.* at 104. As a result, it is Dr. Steinman's opinion that whether or not L.R. had strep in the weeks preceding his hospitalization is irrelevant. *Id.* at 105-06. Dr. Steinman referenced L.R.'s Vanderbilt Medical

Center records, which showed that “ASO titer was negative and anti-DNAase B negative which makes strep sequelae unlikely.” Tr. at 105-06; *see also* Ex. 3 at 2292.

Regarding onset, Dr. Steinman opined that the time between vaccination on August 4th to the time of diagnosis was three weeks and that is consistent with the timeframe between vaccination and neuroinflammation in the case of swine flu vaccine. First Steinman Rep. at 17-18. Dr. Steinman did not initially opine regarding the three to four-day window for onset.

Dr. Steinman opined that L.R.’s leg pain was “thigh pain” that resulted from the number of vaccinations he received, and was not an initial symptom of anti-NMDAR encephalitis. Second Steinman Rep. at 6. Dr. Steinman added that three to four days for a “recall” antibody response was plausible. *Id.* Dr. Steinman additionally stated that “if Petitioner’s theory involved a primary immune response, 3-4 days would be problematic.” *Id.*

In his subsequent reports, Dr. Steinman discussed the three-day onset window. Dr. Steinman stated that a recall response has a logarithmic phase that kicks in three to four days. Second Steinman Rep. at 6-7. The immune system has a lag phase of about seven to ten days however with a subsequent exposure, the lag phase is reduced to one to three days. *Id.* at 7. Dr. Steinman cited to a 2012 IOM report regarding adverse effects of vaccinations as support. *Id.*; *see also* Ex. 12-3 (IOM Report 2012 Adverse Effects Vaccination) (hereinafter “2012 IOM Report”).⁹ The 2012 IOM Report explained that a primary immune response to an antigen contained two phases, a first primary response that has a lag phase, logarithmic phase, and plateau phase. *Id.* at 2. The 2012 IOM report elaborated that it was classically believed that the lag phase longer, between four-to-seven days but is now thought to be one-to-three days and the logarithmic phase occurs over the next three-to-five days. *See id.* The 2012 IOM Report also stated that time periods vary depending on “the route of exposure, the timing of the subsequent exposure, the antigen itself, and the antigen dose.” *Id.*

In response to Dr. Lancaster’s critique of Dr. Steinman’s BLAST searches and E values, Dr. Steinman stated that other filtration methods in his process ensure that the homologies are significant. Third Steinman Rep. at 9-11. Dr. Steinman also added that it is completely unknown whether healthy individuals have anti-NMDA antibodies in their blood or CSF, healthy people are not given spinal taps to see if they have certain antibodies. Tr. at 245-46. Dr. Steinman also clarified that his theory did not rely on whether people have anti-NMDA antibodies latently; L.R. received vaccinations that triggered an immune reaction which produced anti-NMDA antibodies. *Id.* at 247-48.

B. Dr. Eric Lancaster

1. Qualifications

Dr. Lancaster received his Ph.D. in neuroscience and his medical degree from the University of Maryland. Ex. B at 1. Dr. Lancaster completed a neurology residency and

⁹ The Institute of Medicine (now called the National Academy of Medicine) is the medical arm of the National Academy of Sciences and provides advice to the federal government on medical issues.

neuromuscular fellowship at the University of Pennsylvania. *Id.* Dr. Lancaster is an assistant professor of neurology at the University of Pennsylvania and is board certified in neurology and electrodiagnostic medicine, with a special certification in neuromuscular medicine. *Id.* at 2.

Dr. Lancaster's anti-NMDAR encephalitis experience is summarized herein. Dr. Lancaster has written 36 peer-reviewed articles as of February 2017, and most of these papers are entirely or in large part related to anti-NMDAR encephalitis and associated disorders. *Id.* at 4-6; *see also* Ex. A (hereinafter "First Lancaster Rep.") at 1. Dr. Lancaster worked as part of Dr. Dalmau's research team which made "many of the original discoveries in this field." First Lancaster Rep. at 1. Dr. Lancaster also lectures frequently on anti-NMDAR encephalitis and other neurological conditions. *Id.* at 1-2. At his clinic, he treats adult patients with anti-NMDAR encephalitis and related disorders. *Id.* at 2. Through his research, Dr. Lancaster has also studied over 1000 patient samples for NMDAR antibodies. *Id.* Dr. Lancaster directs a quality improvement project to improve the diagnosis of anti-NMDAR encephalitis and related disorders at the University of Pennsylvania. *Id.* Dr. Lancaster treats five to ten new patients per year, and consults on more than 20 cases. Tr. at 142. Dr. Lancaster estimated that he follows a total of 40-50 patients with anti-NMDAR encephalitis. *Id.* His research involves a clinical treatment trial, antibody testing, and generating an animal model to study the disease. *Id.* I recognized Dr. Lancaster as an expert in neurology and anti-NMDAR encephalitis. *Id.* at 143-45.

2. Expert Opinion

Dr. Lancaster was present during both days of the entitlement hearing, testifying on the second day, and has submitted four reports in this case. Ex. A (hereinafter "First Lancaster Rep."), C (hereinafter "Second Lancaster Rep."), D (hereinafter "Third Lancaster Rep."), E (hereinafter "Fourth Lancaster Rep.>").

Dr. Lancaster defined an NMDA (N-methyl-D-aspartate) receptor as a molecule on the surface of cells that detects a neurotransmitter called glutamate. Tr. at 146; First Lancaster Rep. at 15. The antibodies are directly pathogenic and disrupt the functions of the receptor. The antibodies act like a drug that blocks the receptor. First Lancaster Rep. at 18. In anti-NMDAR encephalitis, autoantibodies attack the NMDA receptor, specifically the GluN1 subunit. Tr. at 147. When antibodies attack the receptor, there is less NMDA receptor function, just like when a person takes a drug to block the receptor. *Id.* There is a prodrome¹⁰ in about 70% of cases. *Id.* at 148. Patients develop symptoms of psychosis, abnormal movements, changes in speech, memory, behavior, anger, aggression, and potentially seizures. *Id.* at 148. As the disease progresses, patients develop catatonia, or an inability to respond to the external world; also abnormal movements and unstable blood pressure and heart rates, and eventually can become comatose. *Id.* Patients will recover in the reverse order: from coma to catatonia, to psychosis, improved behavior and cognition. *Id.* The diagnostic of anti-NMDAR encephalitis requires the presence of anti-NMDAR antibodies in spinal fluid. *Id.* at 149.

¹⁰ Prodrome: a premonitory symptom or precursor; a symptom indicating the onset of a disease. DORLAND'S, *Prodrome*, <https://www.dorlandsonline.com/dorland/definition?id=41089> (last accessed on February 8, 2024).

Dr. Dalmau, a leader in the field of NMDAR encephalitis, recently published a paper that found that 412 of 431 patients with NMDAR encephalitis had autoantibodies in both CSF and serum; all patients had antibodies in their CSF. Tr. at 150.

Dr. Lancaster opined that the prodromes preceding the development of anti-NMDAR encephalitis are highly variable; they can be viral, bacterial, and parasitic. Dr. Lancaster stated that “The sheer number of different causes argues against molecular mimicry, since it is nearly impossible to see how all of these infectious agents could mimic the precise 3-dimensional shape of the NMDAR.” Second Lancaster Rep. at 2. Dr. Lancaster wrote, “about half of patients have a “prodrome” illness a week or two before the specific symptoms begin. It is not proven what most of these prodromal infections are or how they cause the illness.” First Lancaster Rep. at 18. For Dr. Lancaster, whether or not L.R. had streptococcus is less important than the fact that he had prodrome prior to the onset of his anti-NMDAR encephalitis, and that illness is more likely causal than Dr. Steinman’s molecular mimicry and recall response theory. Third Lancaster Rep. at 1. Dr. Lancaster opined “the central concept of a recall response is that the patient must have previously developed a specific immune response to the relevant antigen.” *Id.* at 2. “So, [L.R.] might [] have a recall response to vaccine proteins (measles, mumps, etc.) to which he had previously been exposed. However, he could not have a recall response targeting the NMDAR unless he had previously had a significant immune response to the NMDAR.” *Id.* Dr. Lancaster did concede that the current knowledge about anti-NMDAR encephalitis was incomplete, and it is unknown how different infections can trigger the disease, but if we accept the Gautam papers and the theory of molecular mimicry, then “everything would cause everything.” Tr. at 165, 209.

Dr. Lancaster discussed the Gleichman study, which identified the GluN1 subunit as the location where the epitopes must bind in order to trigger anti-NMDAR encephalitis. First Lancaster Rep. at 18; *see also* Gleichman et al., *Anti-NMDA Receptor Encephalitis Antibody Binding Is Dependent on Amino Acid Identity of a Small Region within the GluN1 Amino Terminal Domain*, JOURNAL OF NEUROSCIENCE, August 8, 2012, 32(32):11082–94 (filed as Ex. A, Tab 4) (hereinafter “Gleichman”). The N368/G369 amino acids are critical and the amino acids that bind to this area must be glycosylated for the proper three-dimensional structure to exist. First Lancaster Rep. at 18. This paper suggests that not just any amino acid sequence can bind to this subunit.

A recent study also found that anti-NMDAR encephalitis was reversible. Mice with anti-NMDAR encephalitis, and when injected human NMDAR antibodies demonstrated a reversal of the disease progression. First Lancaster Rep. at 18; *see also* Planaguma et al., *Human N-methyl D-aspartate receptor antibodies alter memory and behaviour in mice*, BRAIN 2014 Nov. 11 (filed as Ex. A, Tab 7).

In his second report, Dr. Lancaster provided his opinion about how anti-NMDAR encephalitis originates. He stated that approximately half of patients have an ovarian teratoma (benign but complex tumor), but some have a preceding HSV encephalitis. Second Lancaster Report at 1. The last group of patients have an infectious prodrome or a definite prior infection. *Id.* at 2. For viral prodromes, Dr. Lancaster opined the typical timeline would be one to two weeks before onset of neurologic symptoms. Tr. at 201. In the case of HSV viral encephalitis, which has a clear association with the HSV virus, the shortest onset window Dr. Lancaster has seen is seven

days, but is more often two to three weeks. *Id.* at 201-02. Dr. Lancaster emphasized that “seven days between stimulus and response would really be the extreme minimum.” *Id.* at 202.

Regarding onset, Dr. Lancaster opined the onset of L.R.’s anti-NMDAR encephalitis was when his leg pain began. First Lancaster Rep. at 16-17. In the Florance paper, limb pain was found to be a disease feature in three patients of the study. *Id.* at 16; *see also* Florance. L.R. also had a prodromal infection a few weeks prior to the onset of his leg pain and neurological symptoms, which is more consistent with the disease process than a three to four day recall response. First Lancaster Rep. at 17. The timing of L.R.’s development of anti-NMDAR encephalitis is more consistent with L.R. getting strep throat in late July, and the onset manifesting approximately two to three weeks later. *Id.* at 21. Dr. Lancaster opined that the timing of when L.R. received his vaccinations, and his onset approximately three days later is “too early for the vaccination[s] to have plausibly caused the autoimmune disease.” *Id.* at 20. Dr. Lancaster opined that an “autoimmune brain disease starts about 7-41 days after the clinical presentation of the viral encephalitis.” *Id.*; *see also* Armangue.

Dr. Lancaster elaborated on L.R.’s leg pain and how it fit the criteria of “limb pain.” Third Lancaster Rep. at 1. Dr. Lancaster described L.R.’s inability to walk, his leg twitching and jerking as a progression of his limb pain. *Id.* At Lincoln Medical Center, the medical records from August 14, 2014 note that L.R. had an unsteady gait and leg pain. *Id.*; *see also* Ex. 2 at 4-5. The Yeshokumar paper found a number of children who presented with gait disturbance as an initial symptom of anti-NMDAR encephalitis, with two children also complaining of leg pain and refusal to bear weight on the affected limb. Third Lancaster Rep. at 1; *see also* Yeshokumar et al., *Gait disturbance as the presenting symptom in young children with anti-NMDA receptor encephalitis*. PEDIATRICS. 2016;138(3):e20160901. doi: 10.1542/peds.2016-0901 (filed as Ex. D, Tab 1) (hereinafter “Yeshokumar”).

Dr. Lancaster disagreed with Dr. Steinman’s opinion that anti-NMDAR encephalitis is a B and T-cell mediated disease; anti-NMDAR encephalitis requires the production of highly specific autoantibodies that must have a three-dimensional structure, which is a B-cell mediated process. First Lancaster Rep. at 21.

Dr Lancaster opined that the linear homology Dr. Steinman found through BLAST searches is not a reliable basis for establishing molecular mimicry and “should be expected to occur by pure random chance.” First Lancaster Rep. at 21-22. Both Silvanovich and Kanduc have found that these short amino acid sequence matches of eight amino acids or fewer have little utility in identifying cross-reactive allergens; further, the number of chance occurrences in shared peptides are “enormous.” *Id.* at 22; *see also* Silvanovich et al., *The value of short amino acid sequence matches for prediction of protein allergenicity*. TOXICOL SCI. 2006;90(1):252-58 (filed as Ex. A, Tab 10); and Kanduc et al., *Massive peptide sharing between viral and human proteomes*. PEPTIDES. 2008;29:1755-66 (filed as Ex. A, Tab 11). Dr. Lancaster also focused on the Gleichman paper which emphasized that NMDAR has a complex three dimensional shape. First Lancaster Rep. at 18. While the N368/G369 amino acids were discovered to be involved with anti-NMDAR encephalitis, Dr. Lancaster also admitted that the site at which anti-NMDAR encephalitis activity was found is not necessarily the only site. Dr. Lancaster emphasized that the site, which involves glycosylation, is important. *Id.*; *see also* Gleichman at 1. Dr. Lancaster added:

The central concept of a recall response is that the patient must have previously developed a specific immune response to the relevant antigen. So, [L.R.] might [sic] certain have a recall response to vaccine proteins (measles, mumps, etc.) to which he had previously been exposed. However, he could not have a recall response targeting the NMDAR unless he had previously had a significant immune response to the NMDAR.

Third Lancaster Rep. at 2.

Regarding Dr. Steinman's assertion that there could be latent NMDAR antibodies in normal people, Dr. Lancaster stated that "We do not observe [NMDAR] antibodies in healthy persons.... Therefore, the idea that Petitioner previously had NMDAR antibodies that could be re-activated is not a tenable explanation in this case." Third Lancaster Rep. at 2. Dr. Lancaster elaborated that in large case series, healthy people do not have meaningful antibodies to NMDAR therefore the idea that L.R. previously had NMDAR antibodies which were re-activated "is not a tenable explanation in this case." Fourth Lancaster Rep. at 1. During his testimony, Dr. Lancaster opined that one in 200 people have any NMDAR antibodies in their serum; this opinion was based on conversations with Dr. Dalmau and testing samples of patients at the University of Pennsylvania. Tr. at 239.

Dr. Lancaster highlighted that Dr. Steinman's three-step filtration process involving BLAST searches and use of the IEDB does not demonstrate any meaningful homology. Fourth Lancaster Rep. at 3. The E-value that Dr. Steinman uses does not effectively filter results. In Dr. Lancaster's search in the IEDB, he found 170 different matches between the measles virus and vaccine antigens. *Id.* at 4. Dr. Lancaster cited to Wiesmüller paper, which showed that patient reactions to the measles vaccine were heterogenous, or that they recognized 7-20% of the sequence. *Id.*; *see also* Wiesmüller et al., *Heterogeneity of linear B cell epitopes of the measles virus fusion protein reacting with late convalescent sera*. J GEN VIROL. 1992;73:2211-2216 (filed as Ex. E, Tab 2). Dr. Lancaster opined that the IEDB filtration is an "empty exercise." Fourth Lancaster Rep. at 4.

VI. Applicable Law

A. Petitioner's Burden in Vaccine Program Cases

Under the Vaccine Act, a petitioner may prevail in one of two ways. First, a petitioner may demonstrate that she suffered a "Table" injury—i.e., an injury listed on the Vaccine Injury Table that occurred within the time period provided in the Table. § 11(c)(1)(C)(i). "In such a case, causation is presumed." *Capizzano v. Sec'y of Health & Hum. Servs.*, 440 F.3d 1317, 1320 (Fed. Cir. 2006); *see* § 13(a)(1)(B). Second, where the alleged injury is not listed in the Vaccine Injury Table, a petitioner may demonstrate that she suffered an "off-Table" injury. § 11(c)(1)(C)(ii).

For both Table and non-Table claims, Vaccine Program petitioners bear a "preponderance of the evidence" burden of proof. Section 13(1)(a). That is, a petitioner must offer evidence that leads the "trier of fact to believe that the existence of a fact is more probable than its nonexistence

before [he] may find in favor of the party who has the burden to persuade the judge of the fact's existence." *Moberly v. Sec'y of Health & Hum. Servs.*, 592 F.3d 1315, 1324 (Fed. Cir. 2010); *see also Snowbank Enter. v. United States*, 6 Cl. Ct. 476, 486 (1984) (mere conjecture or speculation is insufficient under a preponderance standard). Proof of medical certainty is not required. *Bunting v. Sec'y of Health & Hum. Servs.*, 931 F.2d 867, 873 (Fed. Cir. 1991). In particular, a petitioner must demonstrate that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321 (quoting *Shyface v. Sec'y of Health & Hum. Servs.*, 165 F.3d 1344, 1352 (Fed. Cir. 1999)); *Pafford v. Sec'y of Health & Hum. Servs.*, 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner may not receive a Vaccine Program award based solely on his assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. Section 13(a)(1).

In attempting to establish entitlement to a Vaccine Program award of compensation for a non-Table claim, a petitioner must satisfy all three of the elements established by the Federal Circuit in *Althen v. Secretary of Health and Human Services*. 418 F.3d 1274 (Fed. Cir. 2005). *Althen* requires that petitioner establish by preponderant evidence that the vaccinations he received caused her injury "by providing: (1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." *Id.* at 1278.

Each of the *Althen* prongs requires a different showing. Under *Althen* prong one, petitioner must provide a "reputable medical theory," demonstrating that the vaccine received *can cause* the type of injury alleged. *Pafford*, 451 F.3d at 1355-56 (citations omitted). To satisfy this prong, a petitioner's theory must be based on a "sound and reliable medical or scientific explanation." *Knudsen v. Sec'y of Health & Hum. Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Such a theory must only be "legally probable, not medically or scientifically certain." *Id.* at 549.

Petitioner may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *Andreu v. Sec'y of Health & Hum. Servs.*, 569 F.3d 1367, 1378-79 (Fed. Cir. 2009) (citing *Capizzano*, 440 F.3d at 1325-26). Special Masters, despite their expertise, are not empowered by statute to conclusively resolve what are complex scientific and medical questions, and thus scientific evidence offered to establish *Althen* prong one is viewed "not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant evidence standard." *Id.* at 1380. Accordingly, special masters must take care not to increase the burden placed on petitioners in offering a scientific theory linking vaccine to injury. *Contreras v. Sec'y of Health & Hum. Servs.*, 121 Fed. Cl. 230, 245 (2015) ("[p]lausibility ... in many cases may be enough to satisfy *Althen* prong one" (emphasis in original)), *vacated on other grounds*, 844 F.3d 1363 (Fed. Cir. 2017). But this does not negate or reduce a petitioner's ultimate burden to establish her overall entitlement to damages by preponderant evidence. *W.C. v. Sec'y of Health & Hum. Servs.*, 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted).

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1375-77; *Capizzano*, 440 F.3d at 1326 ("medical records and medical opinion

testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a ‘logical sequence of cause and effect show[s] that the vaccination was the reason for the injury’”) (quoting *Althen*, 418 F.3d at 1280). Medical records are generally viewed as particularly trustworthy evidence, because they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec’y of Health & Hum. Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993).

However, medical records and/or statements of a treating physician’s views do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. Section 13(b)(1) (providing that “[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court”); *Snyder v. Sec’y of Health & Hum. Servs.*, 88 Fed. Cl. 706, 746 n.67 (2009) (“there is nothing ... that mandates that the testimony of a treating physician is sacrosanct— that it must be accepted in its entirety and cannot be rebutted”). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should also be weighed against other, contrary evidence also present in the record— including conflicting opinions among such individuals. *Hibbard v. Sec’y of Health & Hum. Servs.*, 100 Fed. Cl. 742, 749 (2011) (not arbitrary or capricious for special master to weigh competing treating physicians’ conclusions against each other), *aff’d*, 698 F.3d 1355 (Fed. Cir. 2012); *Caves v. Sec’y of Health & Hum. Servs.*, No. 06-522V 2011 WL 1935813 at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review den’d*, 100 Fed. Cl. 344, 356 (2011), *aff’d without opinion*, 475 Fed. App’x 765 (Fed. Cir. 2012).

The third *Althen* prong requires establishing a “proximate temporal relationship” between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase “medically acceptable temporal relationship.” *Id.* A petitioner must offer “preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder’s etiology, it is medically acceptable to infer causation.” *de Bazan v. Sec’y of Health & Hum. Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must also coincide with the theory of how the relevant vaccine can cause an injury (*Althen* prong one’s requirement). *Id.* at 1352; *Shapiro v. Sec’y of Health & Hum. Servs.*, 101 Fed. Cl. 532, 542 (2011), *recons. denied after remand on other grounds*, 105 Fed. Cl. 353 (2012), *aff’d without op.*, 503 F. App’x 952 (Fed. Cir. 2013). *Koehn v. Sec’y of Health & Hum. Servs.*, No. 11-355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for review den’d* (Fed. Cl. Dec. 3, 2013), *aff’d*, 773 F.3d 1239 (Fed. Cir. 2014).

B. Law Governing Analysis of Fact Evidence

The process for making factual determinations in Vaccine Program cases begins with analyzing the medical records, which are required to be filed with the petition. Section 11(c)(2). The special master is required to consider “all [] relevant medical and scientific evidence contained in the record,” including “any diagnosis, conclusion, medical judgment, or autopsy or coroner’s report which is contained in the record regarding the nature, causation, and aggravation of the petitioner’s illness, disability, injury, condition, or death,” as well as the “results of any diagnostic or evaluative test which are contained in the record and the summaries and conclusions.” Section

13(b)(1)(A). The special master is then required to weigh the evidence presented, including contemporaneous medical records and testimony. *See Burns v. Sec’y of Health & Hum. Servs.*, 3 F.3d 413, 417 (Fed. Cir. 1993) (it is within the special master’s discretion to determine whether to afford greater weight to contemporaneous medical records than to other evidence, such as oral testimony surrounding the events in question that was given at a later date, provided that such determination is evidenced by a rational determination).

Medical records created contemporaneously with the events they describe are generally trustworthy because they “contain information supplied to or by health professionals to facilitate diagnosis and treatment of medical conditions,” where “accuracy has an extra premium.” *Kirby v. Sec’y of Health & Hum. Servs.*, 997 F.3d 1378 (Fed. Cir. 2021) citing *Cucuras*, 993 F.2d at 1528. This presumption is based on the linked proposition that (i) sick people visit medical professionals; (ii) sick people honestly report their health problems to those professionals; and (iii) medical professionals record what they are told or observe when examining their patients in as accurate a manner as possible, so that they are aware of enough relevant facts to make appropriate treatment decisions. *Sanchez v. Sec’y of Health & Hum. Servs.*, No. 11-685V, 2013 WL 1880825 at *2 (Fed. Cl. Spec. Mstr. Apr. 10, 2013) *mot. for rev. denied*, 142 Fed. Cl. 247, 251-52 (2019), *vacated on other grounds and remanded*, 809 Fed. Appx. 843 (Fed. Cir. Apr. 7, 2020).

Accordingly, if the medical records are clear, consistent, and complete, then they should be afforded substantial weight. *Lowrie v. Sec’y of Health & Hum. Servs.*, No. 03-1585V, 2005 WL 6117475 at *20 (Fed. Cl. Spec. Mstr. Dec. 12, 2005). Indeed, contemporaneous medical records are generally found to be deserving of greater evidentiary weight than oral testimony— especially where such testimony conflicts with the record evidence. *Cucuras*, 993 F.2d at 1528; see also *Murphy v. Sec’y of Health & Hum. Servs.*, 23 Cl. Ct. 726, 733 (1991), *aff’d per curiam*, 968 F.2d 1226 (Fed. Cir. 1992), *cert. den’d*, *Murphy v. Sullivan*, 506 U.S. 974 (1992) (citing *United States v. U.S. Gypsum Co.*, 333 U.S. 364, 396 (1947) (“[i]t has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight.”)).

However, there are situations in which compelling oral testimony may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. *Campbell v. Sec’y of Health & Hum. Servs.*, 69 Fed. Cl. 775, 779 (2006) (“like any norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking”); *Lowrie*, 2005 WL 6117475 at *19 (“[w]ritten records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent”) (quoting *Murphy*, 23 Cl. Ct. at 733)). Ultimately, a determination regarding a witness’s credibility is needed when determining the weight that such testimony should be afforded. *Andreu*, 569 F.3d at 1379; *Bradley v. Sec’y of Health & Hum. Servs.*, 991 F.2d 1570, 1575 (Fed. Cir. 1993).

When witness testimony is offered to overcome the presumption of accuracy afforded to contemporaneous medical records, such testimony must be “consistent, clear, cogent and compelling.” *Sanchez*, 2013 WL 1880825 at *3 (citing *Blutstein v. Sec’y of Health & Hum. Servs.*, No. 90-2808V, 1998 WL 408611 at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)). In determining the accuracy and completeness of medical records, the Court of Federal Claims has listed four possible explanations for inconsistencies between contemporaneously created medical records and later

testimony: (1) a person's failure to recount to the medical professional everything that happened during the relevant time period; (2) the medical professional's failure to document everything reported to her or him; (3) a person's faulty recollection of the events when presenting testimony; or (4) a person's purposeful recounting of symptoms that did not exist. *LaLonde v. Sec'y of Health & Hum. Servs.*, 110 Fed. Cl. 184, 203-04 (2013), *aff'd*, 746 F.3d 1334 (Fed. Cir. 2014). In making a determination regarding whether to afford greater weight to contemporaneous medical records or other evidence, such as testimony at hearing, there must be evidence that this decision was the result of a rational determination. *Burns*, 3 F.3d at 417.

C. Analysis of Expert Testimony

Establishing a sound and reliable medical theory connecting the vaccine to the injury often requires a petitioner to present expert testimony in support of his or her claim. *Lampe v. Sec'y of Health & Hum. Servs.*, 219 F.3d 1357, 1361 (Fed. Cir. 2000). Vaccine Program expert testimony is usually evaluated according to the factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 594-96 (1993). See *Cedillo v. Sec'y of Health & Hum. Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec'y of Health & Hum. Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999)). "The *Daubert* factors for analyzing the reliability of testimony are: (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community." *Terran*, 195 F.3d at 1316 n.2 (citing *Daubert*, 509 U.S. at 592-95).

The *Daubert* factors play a slightly different role in Vaccine Program cases than they do when applied in other federal judicial fora. *Daubert* factors are employed by judges to exclude evidence that is unreliable and potentially confusing to a jury. In Vaccine Program cases, these factors are used in the weighing of the reliability of scientific evidence. *Davis v. Sec'y of Health & Hum. Servs.*, 94 Fed. Cl. 53, 66-67 (2010) ("uniquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted"). The flexible use of the *Daubert* factors to evaluate persuasiveness and reliability of expert testimony has routinely been upheld. See, e.g., *Snyder*, 88 Fed. Cl. at 743. In this matter, (as in numerous other Vaccine Program cases), *Daubert* has not been employed at the threshold, to determine what evidence should be admitted, but instead to determine whether expert testimony offered is reliable and/or persuasive.

Respondent frequently offers one or more experts of his own in order to rebut a petitioner's case. Where both sides offer expert testimony, a special master's decision may be "based on the credibility of the experts and the relative persuasiveness of their competing theories." *Broekelschen v. Sec'y of Health & Hum. Servs.*, 618 F.3d 1339, 1347 (Fed. Cir. 2010) (citing *Lampe*, 219 F.3d at 1362). However, nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Snyder*, 88 Fed. Cl. at 743 (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997)). A "special master is entitled to require some indicia of reliability to support the assertion of the expert witness." *Moberly*, 592 F.3d at 1324. Weighing the relative persuasiveness of competing expert testimony, based on a

particular expert's credibility, is part of the overall reliability analysis to which special masters must subject expert testimony in Vaccine Program cases. *Id.* at 1325-26 (“[a]ssessments as to the reliability of expert testimony often turn on credibility determinations”); *see also Porter v. Sec’y of Health & Hum. Servs.*, 663 F.3d 1242, 1250 (Fed. Cir. 2011) (“this court has unambiguously explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act”).

D. Consideration of Medical Literature

Finally, although this decision discusses some but not all of the medical literature in detail, I have reviewed and considered all of the medical records and literature submitted in this matter. *See Moriarty v. Sec’y of Health & Hum. Servs.*, 844 F.3d 1322, 1328 (Fed. Cir. 2016) (“We generally presume that a special master considered the relevant record evidence even though [s]he does not explicitly reference such evidence in h[er] decision.”); *Simanski v. Sec’y of Health & Hum. Servs.*, 115 Fed. Cl. 407, 436 (2014) (“[A] Special Master is ‘not required to discuss every piece of evidence or testimony in her decision.’” (citation omitted)), *aff’d*, 601 F. App’x 982 (Fed. Cir. 2015).

VII. Analysis

Because Petitioner does not allege an injury listed on the Vaccine Injury Table, her claim is classified as “off-Table.” As noted above, to prevail on an “off-Table” claim, Petitioner must prove by preponderant evidence that L.R. suffered an injury and that this injury was caused by the vaccinations at issue. *See Capizzano*, 440 F.3d at 1320. The parties agree that L.R. was correctly diagnosed with anti-NMDAR encephalitis. Joint Prehearing Submissions at 2.

A. Althen Prong One

In the context of the Program, “to establish causation, the standard of proof is preponderance of evidence, not scientific certainty.” *Langland v. Sec’y of Health & Hum. Serv.*, 109 Fed. Cl. 421, 441 (2013). Petitioner’s burden under *Althen*’s first prong is to provide a medical theory causally connecting the vaccination and the injury. *Id.* This theory must be sound and reliable. *Boatmon*, 941 F.3d at 1359.

Petitioner contends that the MMR and/or the varicella vaccines caused L.R. to develop anti-NMDAR encephalitis via molecular mimicry. Dr. Steinman posits that there is homology between components of the MMR and varicella vaccines and the NMDA receptor; the homologies between the vaccines and the NMDA receptor triggered an autoimmune response where the antibodies generated as a response to vaccination attack NMDA receptors, which leads to the development of anti-NMDAR encephalitis.

The first part of Dr. Steinman’s theory involves a BLAST search to see if there are homologies between the components of the vaccines and the NMDA receptor. BLAST, or Basic Local Alignment Search Tool, is a program that “finds regions of similarity between biological sequences.” NIH, National Library of Medicine, BLAST; blast.ncbi.nlm.nih.gov/Blast.cgi; Tr. at 79. Dr. Steinman, through a BLAST search, identified homologies, or what Dr. Steinman believes

are “relevant molecular mimics” between the MMR and varicella vaccines and the NMDA receptors, thus linking the vaccines to anti-NMDAR encephalitis. First Steinman Rep. at 6. Dr. Steinman defined a “relevant molecular mimic” as “a run of 5 or more of 12 amino acids that are identical.” *Id.*

Dr. Steinman bases his position that five or more identical amino acids will produce disease on three main papers. These papers, each authored by Gautam and Dr. Steinman himself, address the question of how much homology between a self-antigen and a foreign antigen is enough to induce autoimmunity. The Gautam papers found that five identical amino acids out of 12 could trigger neuroinflammation, in the form of experimental autoimmune encephalomyelitis (EAE; the animal model of MS). The amino acids only need to be in identical locations and do not have to be in consecutive order. *See* Gautam 1. According to Dr. Steinman, the three papers collectively demonstrate that a sequence of five out of 12 amino acids is sufficient to lead to neurologic disease. Tr. at 30; (citing Gautam 1; Gautam et al., *Minimum structural requirements for peptide presentation by major histocompatibility complex class II molecules: Implications in induction of autoimmunity*, 161 PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES USA, 767-771 (1994) (filed as Ex. 11, Tab 15) (hereinafter “Gautam 2”); Gautam et al., *A polyalanine peptide containing only five native myelin basic protein residues induces autoimmune encephalomyelitis*, 127 JOURNAL OF EXPERIMENTAL MEDICINE 605-609 (1992) (filed as Ex. 11, Tab 16) (hereinafter “Gautam 3”)). This is the second part of Dr. Steinman’s filtration process, limiting BLAST search results to what is a “relevant significant homology.” First Steinman Rep. at 6.

With respect to the BLAST searches in this case, Dr. Steinman noted that the sequence “QYPPTFRFG” from the rubella virus is structurally similar to “QYPPFRFG” from the NMDA receptor. First Steinman Rep. at 11. Through his BLAST search, Dr. Steinman identified a 7 of 8 amino acid homology in this region (QYPP_RFG). *Id.* Dr. Steinman opined that the Gautam papers demonstrate that this is sufficient to induce autoimmunity via the mechanism of molecular mimicry. Dr. Steinman also identified another homologous sequence between the NMDA receptor and the rubella vaccine (VRGVW_); two relevant homologous sequences between the mumps virus and NMDA receptor (_N_ _ _I_PRG) and (ST_R_ _RS_Y_D), and (RP_ _ _E_PDP); three relevant homologous sequences between the measles virus and the NMDA receptor (A_ _ _ _YD_YL), (FV_ _ _SP_P), and (V_ _ _L_ _GHA_F); and one relevant homologous sequence between the varicella virus and the NMDA receptor (TPK_ _G_ _ _Q). *Id.* at 11-17.

While molecular mimicry is a theory that is generally accepted in the Vaccine Program, a “simple invocation of the term generally does not carry a petitioner’s burden of proof.” *Deshler v. Sec’y of Health & Hum. Servs.*, No. 16-1070V, 2020 WL 4593162, at *20 (Fed. Cl. Spec. Mstr. July 1, 2020) (citing *Forrest v. Sec’y of Health & Hum. Servs.*, No. 14-1046V, 2019 WL 925495, at *3 (Fed. Cl. Spec. Mstr. Jan. 18, 2019)). This is in part because “the finding of sequence homology does not necessarily mean the similarity has significance to the immune system.” *Tullio v. Sec’y of Health & Hum. Servs.*, No. 15-51V, 2019 WL 7580149, at *15 (Fed. Cl. Spec. Mstr. Dec. 19, 2019), *mot. for rev. denied*, 149 Fed. Cl. 448 (2020); *see also Caredio v. Sec’y of Health & Hum. Servs.*, No. 17-0079V, 2021 WL 4100294, at *31 (Fed. Cl. Spec. Mstr. July 30, 2021), *mot. for rev. denied*, 2021 WL 6058835 (2021) (“demonstration of homology alone is not enough to establish a preponderant causation theory”) (citing *Schultz v. Sec’y of Health & Hum. Servs.*, No. 16-539V, 2020 WL 1039161, at *22 n. 24 (Fed. Cl. Spec. Mstr. Jan. 24, 2020) (“[m]ere

demonstration of theoretical homology alone, based on computer-driven searches involving databases of amino acid sequences, does not carry the day”)).

Dr. Lancaster opined that the numerous “relevant molecular mimics” Dr. Steinman found, listed above, highlight the flaws in Dr. Steinman’s molecular mimicry theory and approach. Dr. Lancaster stated that the longest consecutive sequence of homology is three, which “is a trivial sort of homology and inevitable when comparing any two large proteins.” First Lancaster Rep. at 23. As a comparison, Dr. Lancaster ran a BLAST search looking for homology between a common chicken protein and the NMDAR subunit used by Dr. Steinman (GRIN2C Q14957) and found the sequence AF_R_L_N, and a sequence of homology between dystrophin, a common protein in chicken muscle and the GRIN2C subunit, G_TVAV. *Id.* at 24-25. These sequences would have sufficient homology to trigger an immune reaction according to Dr. Steinman and the Gautam papers. *Id.* at 24.

Dr. Lancaster contested the usefulness of these sequences in his expert report. He noted that “the ‘NMDA receptor’ is not one single thing. Rather multiple different types of NMDA receptors can be made from different subunits.” First Lancaster Rep. at 23. There is a consensus among the leading anti-NMDAR encephalitis researchers that the NMDA receptor has a complex three-dimensional shape, yet it has been discovered that the GluN1/GRIN1 subunit where the antibodies attach, causes a loss of NMDAR function “acting something like a drug that blocks the receptor.” *Id.* at 18. Dr. Steinman ran BLAST searches for homologies between the measles, mumps, rubella, and varicella viruses against AAA88096.1,¹¹ which is the NMDA receptor GRIN2C subunit. *Id.* at 23. Dr. Lancaster noted that “there is not any human disease associated with GRIN2C antibodies.” *Id.*

Dr. Lancaster highlighted the Gleichman article to provide additional context to Dr. Steinman’s BLAST searches and their lack of relevance to the case at hand. The Gleichman paper extensively tested sites where antibodies could bind to the NMDA receptor, which would trigger anti-NMDAR encephalitis, and identified N368/G369 on the GluN1/GRIN1 subunit of the anti-NMDA receptor as a region where antibodies bind. Gleichman at 11082.

The limitations of the Gleichman paper are discussed in a subsequently published book chapter, which Gleichman co-authored, on anti-NMDAR encephalitis published in 2018. Lynch, et al., *Anti-NMDA Receptor Encephalitis: Clinical Features and Basic Mechanisms*, ADVANCES IN PHARMACOLOGY, Chapter 11, 2018 (filed as Ex. 12, Tab 10) (hereinafter “Lynch chapter”). In the chapter, the authors state:

Development of immunoreactivity to PtAbs is blocked by tunicamycin, an agent altering glycosylation of proteins, and removal of a single glycosylation site (N368) removes the epitope of GluN1. However, alternative mutations that remove glycosylation but not N368 still retain the ability to be recognized by PtAbs. Thus, multiple mutations in the region around N368 remove epitope recognition, but they

¹¹ National Library of Medicine, *NMDA reception [Homo sapiens] AAA88096.1*, NIH National Center for Biotechnology Information, <https://www.ncbi.nlm.nih.gov/protein/AAA88096.1> (last accessed March 20, 2024).

do not correlate perfectly with glycosylation, suggesting that this region creates a conformation of GluN1 that is necessary for generating epitope recognition. It also does not prove that this region is sufficient for immunoreactivity; the smallest portion of GluN1 that still generates immunoreactivity includes the full N-terminal domain linked to a transmembrane region, a much larger region than that which is necessary. These data are most easily interpreted as showing that GluN1 contains a conformational epitope in the first 381 amino acids of GluN1 that requires the region near amino acid 368 to develop proper conformation.

Lynch chapter at 244-45. Dr. Steinman highlighted this text, noting that “the exact nature of the conformational epitope in NMDA-R encephalitis is still not very clear...” Second Steinman Rep. at 18. However, the Lynch chapter solely discusses the involvement of the GluN1 subunit. In the section within the Lynch chapter, entitled “Basic Mechanisms of Anti-NMDARE,” the authors state, “Biochemically, the site of epitope recognition for the antibodies of anti-NMDARE is on GluN1.” Lynch chapter at 244. The Lynch chapter does not state that the GRIN2C subunit is involved in disease. Furthermore, in a paper proffered by Dr. Steinman (Ex. 11.9), researchers studied the NR1 and NR2 subunits, and discovered “the main epitope region recognised by all patients’ antibodies lies within the extracellular region of the NR1 subunit.” Dalmau et al., *Anti-NMDA-receptor encephalitis: case series and analysis of the effects of antibodies*. LANCET NEUROLOGY 2008 December; 7(12): 1091–1098 (filed as Ex. 11.9). Dr. Steinman has not provided any evidence that the NR2 or GRIN2C subunit is involved with anti-NMDAR encephalitis.

Dr. Steinman described the third step in his process as an additional filtration step, thus “increas[ing] the stringency” in the search for molecular mimics. Third Steinman Rep. at 10. In support of this step, Dr. Steinman proffered additional results from the IEDB to demonstrate how these homologies were significant. Dr. Steinman testified that the IEDB is a government database funded by the National Institute of Allergy and Infectious Disease, which provides a list of all epitopes in peer-reviewed literature. Tr. at 90, 94. Dr. Steinman inputted the BLAST search homologies listed above into IEDB to see if there was any literature featuring these sequences, thus demonstrating that the mimics were significant in that they elicited a response from the human immune system. Dr. Steinman was able to identify two results for the sequences of LHPGHALF and FVIVESPDP in the IEDB. Third Steinman Rep. at 5-6. The first sequence, LHPGHALF, was found in association with the HLA B*27 gene while the other sequence was a known epitope for Influenza A hemagglutinin. Dr. Steinman did not discuss how the HLA B*27 gene or how influenza A relate to anti-NMDAR encephalitis, except to note that “These epitopes have been described on the IEDB. They help to show that indeed there are homologies between the Edmonston strain measles antigen in the MMR vaccine and NMDA-R, that have been described on the IEDB, and that these epitopes homologies with other viral and other protein antigens. Such epitopes are recognized by the human immune system and therefore they could indeed be recognized by [L.R.]’s immune system following vaccination.” Third Steinman Rep. at 10.

Dr. Lancaster disagreed that this third filtration step added anything significant to the analysis of the causal theory. He noted that most of the vaccine protein sequences are studied and thus end up in the IEDB; as a result, this additional step in Dr. Steinman’s process doesn’t narrow things down at all. Tr. at 174-75. It “doesn’t suddenly make random stretches of amino acid

homology significant predictors of autoimmunity.” *Id.* at 175. Based on the above, I do not find these searches persuasive in supporting Dr. Steinman’s molecular mimicry theory.

In summary, Petitioner has demonstrated there are short sequence homologies between portions of the vaccines L.R. received and a portion of the NMDA receptor not known to be associated with disease. Dr. Steinman’s third step in his filtration process does not persuasively render the proffered sequence homologies relevant to the initiation of L.R.’s condition. Therefore, I find that Petitioner has not presented preponderant evidence that the MMR and/or varicella vaccines can cause anti-NMDAR encephalitis. Further, Dr. Lancaster who has a specialty in treating anti-NMDAR encephalitis and has been involved in the research on the condition with leading authorities on the disease was more persuasive than Dr. Steinman in this case. Petitioner has not satisfied the first *Athen* prong.

C. *Athen* Prong Two

Under *Athen*’s second prong, a petitioner must “prove a logical sequence of cause and effect showing that the vaccination was the reason for the injury.” *Athen*, 418 F.3d at 1278. The sequence of cause and effect must be “‘logical’ and legally probable, not medically or scientifically certain.” *Id.* A petitioner is not required to show “epidemiologic studies, rechallenge, the presence of pathological markers or genetic disposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect.” *Id.* (omitting internal citations). *Capizzano v. Sec’y of Health & Hum. Servs.*, 440 F.3d 1317, 1325 (Fed. Cir. 2006). Instead, circumstantial evidence and reliable medical opinions may be sufficient to satisfy the second *Athen* prong. *Isaac v. Sec’y of Health & Hum. Servs.*, No. 08-601V, 2012 WL 3609993, at *25 (Fed. Cl. Spec. Mstr. July 30, 2012), *mot. for rev. denied*, 108 Fed. Cl. 743 (Fed. Cl. 2013), *aff’d*, 540 Fed. Appx. 999 (Fed. Cir. 2013).

1. The Existence of Low Level NMDAR Antibodies at the time of Vaccination

When discussing an appropriate temporal interval pursuant to *Athen* prong three, Dr. Steinman initially opined that L.R. was *diagnosed* with anti-NMDAR encephalitis three weeks after vaccination. First Steinman Rep. at 1, 4, 17. He opined that this period of time was consistent with neuroinflammation caused by the swine flu vaccine. *Id.* at 17-18. Dr. Steinman did not discuss the issue of timing with respect to the *onset* of L.R.’s condition. *See generally*, First Steinman Rep. Dr. Lancaster submitted an expert report responding to Dr. Steinman’s opinion. First Lancaster Rep. In this report, Dr. Lancaster pointed out this discrepancy, and opined that three to four days, the time between vaccination and onset of L.R.’s condition, was “too early for the vaccination to have plausibly caused the autoimmune disease.” First Lancaster Rep. at 20.

After this first round of expert reports, I asked the experts several questions. In particular, I posed the following question to Dr. Steinman:

Dr. Lancaster states that [] L.R.’s onset of psychiatric symptoms began soon after vaccine administration, August 8, 2014 at the latest. Do you believe a period of three to four days is medically feasible for the development of an antibody-mediated immune response? Why or why not?

Scheduling Order dated July 1, 2019; ECF No. 35. Dr. Steinman responded, “A period of 3 to 4 days is medically feasible for a “recall” antibody response. If Petitioner’s theory involved a primary immune response, 3 to 4 days would be problematic.” Second Steinman Rep. at 6.

Dr. Lancaster testified that based on his work in his own lab and on his conversations with Dr. Dalmau, he personally sees fewer than 1 in 200 healthy patients who have very low levels of NMDAR antibodies in their serum. Tr. at 237-40. He stated that a few people might put the number higher, at 1 in 100. *Id.* at 240.

Dr. Steinman clarified at the entitlement hearing that Petitioner’s causal theory assumes the existence of low level NMDAR antibodies at the time of L.R.’s vaccination.

Q. You testified about recall response in this particular case and that the initial vaccination that L. received resulted in the production of some antibodies and that ... these antibodies can and did exist at a low enough level such that they didn’t cause disease. Is that correct?

A. That’s what I testified, and that would be the explanation that time one, the level went up to X, time two it went up to higher at some point with a boost. ... So I think that that idea has good foundations, that it took that vaccine in early August to boost his immunity at a level that he became clinically affected by NMDA receptor antibodies.

Tr. at 131-32. Dr. Lancaster responded that it is improbable and highly speculative that L.R. had “NMDA antibodies [that] were there sort of hidden, generated at just the right level that they didn’t cause disease.” Tr. at 187.

Although the testimony of Dr. Lancaster establishes that between .5% and 1% of people without disease have low levels of NMDAR antibodies in their serum, there is no evidence that L.R. had these antibodies at the time of vaccination. In *Boatmon v. Secretary of Health and Human Services*, the Federal Circuit rejected the special master’s finding that because 50-70% of SIDS cases have a brainstem abnormality, the child in the *Boatmon* case likely had one as well, absent any actual evidence that he did. *Boatmon*, 941 F.3d at 1362-63. The court in *Boatmon* reiterated its finding in *Knudsen*, that statistical evidence alone cannot constitute proof of actual causation. *Id.* at 1363; citing *Knudsen*, 35 F.3d at 550. The case at bar is not even supported by statistics.

The existence of a brainstem abnormality was a central component of petitioners’ theory involving the “Triple Risk Model” in *Boatmon*. Similarly, the existence of NMDAR antibodies at the time of L.R.’s vaccination must be assumed in order for Petitioner to prevail. The lack of any evidence on this point prevents me from doing so. If L.R. did not have NMDAR antibodies at the time of vaccination, he necessarily experienced a primary immune response, which Dr. Steinman opined would not likely occur in three to four days (“If Petitioner’s theory involved a primary immune response, 3 to 4 days would be problematic.”). Second Steinman Rep. at 6.

2. Strep Throat Infection

L.R.'s medical records consistently document that he had a strep throat infection at the end of July, 2014. *See, e.g.* Ex. 3 at 41 (medical record from August 9, 2014, stating that L.R. "had strep throat two weeks ago, and has recovered completely from that."); Ex. 3 at 2262 (August 15, 2014 medical record documenting that L.R. "was in his usual state of good health until 3 weeks ago when he developed strep throat."); Ex. 3 at 2391 (neurology note dated August 24, 2014, stating "[patient] had a strep throat infection 3 weeks prior to presentation. This was diagnosed via rapid strep test and he was treated with a course of amoxicillin."). Ex. 3 at 2271 (medical record from August 16, 2014 noting "multiple family members with strep throat"). Ex. 3 at 2246 (neurology consultation note from August 15, 2014 documenting "known strep exposure and infection.").

Petitioner further corroborated this point in her affidavit, where she averred that sometime in July of 2014, L.R. had strep throat infection, was prescribed antibiotics, and recovered quickly after that. Aff. at 1. During trial, Petitioner confirmed that L.R. complained that his throat hurt, she took him to the doctor, and the doctor prescribed a ten day course of amoxicillin. Tr. at 8. L.R. felt better within a couple of days, but he took the ten-day course of antibiotics as prescribed. *Id.*

On August 18, 2014, L.R.'s ASO and anti-DNase-B tests returned as negative. Ex. 3 at 2292. As a result Dr. Steinman testified that he did not believe that strep throat was the cause of L.R.'s anti-NMDAR encephalitis. Tr. at 104-06. Dr. Lancaster disagreed with Dr. Steinman's position. He testified as follows.

Those tests refer to specific measurements of high antibody levels to strep, and particularly I think that they were talking about this idea of Sydenham chorea which Dr. Steinman discussed yesterday, which is a very specific syndrome of abnormal movements occurring after [...] streptococcal infections, where it's thought that the antibodies can even cross-react against a part of the brain called the basal ganglia and cause movement symptoms. They did not think that L. had Sydenham chorea. I agree that it's correct that he did not have that. I think that the lack of these antibodies doesn't really tell us anything about the preceding infection of a strep throat triggering anti-NMDA receptor encephalitis, which is a separate question, and we don't require any particular, you know, type of streptococcal antibodies or even any particular one infection on these prodromal illnesses...

Tr. at 204-05. Petitioner did not address this point further, either during cross examination of Dr. Lancaster,¹² or during rebuttal testimony from Dr. Steinman.

The underlying medical record documenting the strep throat was not filed. However, the other medical records which detail that L.R. did have this infection are persuasive. This fact was consistently reported to medical professionals in order to appropriately treat L.R. In this context, such statements are considered reliable. *Cucuras*, 993 F.2d at 1528 ("Medical records, in general,

¹² Petitioner's only question on the topic was to ask whether Dr. Lancaster agreed that L.R.'s ASO and anti-DNase tests were negative. Tr. at 211.

warrant consideration as trustworthy evidence. The records contain information supplied to or by health professionals to facilitate diagnosis and treatment of medical conditions. With proper treatment hanging in the balance, accuracy has an extra premium.”) Further, the records document more than just the existence of the infection itself, but also describe corroborating circumstances, such as the fact that multiple family members had strep throat, that L.R. had a positive rapid strep test, and that he was prescribed and completed a 10-day course of the antibiotic amoxicillin. Based on the above, I conclude there is preponderant evidence that L.R. had a strep throat infection at the end of July, 2014.

Dalmau estimates that approximately 70% of patients diagnosed with anti-NMDAR encephalitis have prodromal infectious symptoms. Dalmau at 2. Yeshokumar et al. note that the pathophysiology of anti-NMDAR encephalitis in children is postinfectious autoimmune. Yeshokumar et al., *Gait Disturbance as the Presenting Symptom in Young Children With Anti-NMDA Receptor Encephalitis*, 138 PEDIATRICS 3, 1-8, Table 5 (2016) (filed as Ex. D, Tab 1). Cai et al. describe specific infections that have preceded anti-NMDAR encephalitis. They state, “Previous reports have suggested that anti-NMDAR encephalitis may be associated with the herpes simplex virus, mycoplasma pneumoniae, measles virus, mumps, and group A hemolytic streptococcus.” Cai et al., *Anti-N-methyl-D-aspartate receptor encephalitis associated with acute Toxoplasma gondii infection*, 97 MEDICINE 7, 1-4 (2018) (filed as Ex. C, Tab 4). In referencing this literature, Dr. Lancaster opined that “We have every reason to believe that infections can cause anti-NMDAR encephalitis in general. Streptococcus is among the reported triggers.” Second Lancaster Rep. at 2; Tr. at 158. Dr. Lancaster testified that L.R.’s strep throat infection a couple of weeks before onset of anti-NMDAR encephalitis fits within the expected latency period to attribute causation to the virus; and further, that it is “a much better fit in terms of a plausible latency than four days.” Tr. at 204. Although Dr. Lancaster concluded that the strep infection most likely caused L.R. to develop anti-NMDAR encephalitis, he could not describe a specific mechanism for how this happened. Tr. at 209-10.

Dr. Lancaster also emphasized that while most patients with anti-NMDAR encephalitis have a viral prodrome, these illnesses are frequently not diagnosed with specificity. He testified:

Often we just don’t know what it was. The child or the adult reported diarrhea for a few days two weeks ago, and that’s the information we have, and no tests were done, or the child had an upper respiratory infection two or three weeks ago, called a [cough] or a sore throat, and that’s what we have.

Tr. at 205. Accordingly, while I do find there is preponderant evidence that L.R. had strep throat approximately two to three weeks before onset of his anti-NMDAR encephalitis, the specificity of the viral prodrome is less significant than the fact that there was a viral prodrome.

I acknowledge that Petitioner is not required to eliminate other potential causes of anti-NMDAR encephalitis in order to be entitled to compensation in the Vaccine Program. *See Walther v. Sec’y of Health & Hum. Servs.*, 485 F.3d 1146, 1149-52 (Fed. Cir. 2007) (concluding a petitioner does not bear the burden of eliminating alternative independent potential causes). However, I find it appropriate to consider other possible sources of injury in making a determination pursuant to *Althen* prong two. *Stone v. Sec’y of Health & Hum. Servs.*, 676 F.3d 1373, 1379-80 (Fed. Cir.

2012); *see also Winkler v. Sec’y of Health & Hum. Servs.*, 88 F.4th 958, 963 (Fed. Cir. 2023). L.R.’s prodromal viral illness, more likely than not strep throat, is a possible source of his injury. The existence of this other source of injury weakens the persuasiveness of Petitioner’s prong two showing in that it reduces the likelihood of the vaccine’s causal role.

The Federal Circuit in *Capizzano* noted that “[t]he second prong of the *Althen* ... test is not without meaning.” *Capizzano*, 440 F.3d at 1327. In this case, Petitioner has not presented evidence supporting her position that L.R.’s vaccines did cause him to develop anti-NMDAR encephalitis. Instead, in order for her causal theory to be viable, Petitioner requires I assume that L.R. had low level NMDAR antibodies at the time of vaccination, where there is no evidence of such. Further, she alleges a logical sequence of cause and effect when L.R.’s strep throat infection constitutes another source of injury. Based on the above, Petitioner has failed to preponderantly demonstrate that L.R.’s vaccinations “did cause” his medical condition, and has thus not established the second prong of *Althen*.

D. *Althen* Prong Three

The timing prong contains two parts. First, a petitioner must establish the “timeframe for which it is medically acceptable to infer causation” and second, she must demonstrate that the onset of the disease occurred in this period. *Shapiro v. Sec’y of Health & Hum. Servs.*, 101 Fed. Cl. 532, 542-43 (2011), *recons. denied after remand on other grounds*, 105 Fed. Cl. 353 (2012), *aff’d without op.*, 503 F. App’x 952 (Fed. Cir. 2013).

1. Onset of Anti-NMDAR Encephalitis

Petitioner contends that the onset of L.R.’s anti-NMDAR encephalitis was three to four days post vaccination and started with L.R.’s nightmares and altered mental state. Respondent argues that onset began with L.R.’s thigh pain immediately after vaccination on August 4, 2014.

It is uncontested that L.R. began to hallucinate at around 11:00pm on August 7, 2014, approximately three and one half days after vaccination. *See, e.g.*, Ex. 2 at 1-3 (Lincoln Medical Center ER record documenting altered mental status). Respondent does not contest this altered mental state was caused by anti-NMDAR encephalitis. The analysis below discusses thigh pain and whether that constitutes the beginning of L.R.’s condition.

First, I note that Respondent has presented evidence that limb pain can be an initial symptom of anti-NMDAR encephalitis. Florance et al. observe that some anti-NMDAR encephalitis patients they studied experienced early symptoms of limb pain. Florance at 3. Yeshokumar et al. describe children with anti-NMDAR encephalitis who presented with gait disturbance as their initial symptom. Two of these children refused to bear weight on the leg, one was described as experiencing intermittent leg pain. Yeshokumar at 2, 3. Dr. Lancaster also testified persuasively on this subject. Tr. at 224-27. While leg pain and gait disturbance are not the typical presentation of patients with anti-NMDAR encephalitis, medical literature documents this can occur.

L.R. received five vaccines injected into his thighs on August 4, 2014. Petitioner testified that L.R. complained about his legs hurting after vaccination. Tr. at 11. Petitioner described that L.R.'s leg pain lasted three to four days. *Id.* at 59. Additionally, L.R.'s legs remained red and swollen a couple of days after vaccination. *Id.* at 59-60. Petitioner had to put cold rags on his thighs because of the heat, the redness, and the swelling. *Id.* at 58.

The medical records generally support this testimony. On August 8, 2014, Dr. Cline documented that "The patient is a four-year-old male seen in clinic three days ago for a well child check and had not had any medical issues at that time. Patient received standard four-year-old injections in bilateral thighs, and since that time, patient has been complaining intermittently of severe thigh pains." Ex. 18 at 1. The next day, on August 9, 2014, the medical records note that "Patient with intermittent complaints of muscle pains, especially in the thighs for the past week since getting immunization shots. Patient will complain of discomfort for an hour and then it will resolve and return sometime later." *Id.* at 10.

On August 14, 2014 at 2030, L.R. presented to the Lincoln ED. The ED triage note documents the chief complaint as "uncontrollable movements of arms and legs." The record further notes that L.R. "had immunizations on Monday and these movements started on Thursday." Ex 18 at 19. L.R. was carried to the ER because his legs would buckle under him. *Id.* At 2240, the ED record documents that L.R. "was crying at night [because of] bilateral leg pain." Ex. 2 at 4. The note further summarizes that "[Patient] now continues with twitching and jerking movements of bilateral legs and [is] unable to walk." *Id.* The onset of twitching was noted to be gradual over the past two days. *Id.*

Dr. Lancaster opined that the length of time that L.R. experienced leg pain indicates that this pain constituted the beginning of anti-NMDAR encephalitis. He testified: "If this were just local pain or inflammation from the vaccine, this would have rapidly faded away." Tr. at 191. Dr. Lancaster further testified that the evolution of the leg pain into difficulty walking also supported his position that the pain was the onset of the anti-NMDAR encephalitis. *Id.*

Notably, however, L.R. received five vaccines injected into his thighs. Petitioner credibly testified that L.R.'s thighs were red, swollen, and hot for a couple of days after vaccination. Tr. at 59-60. No evidence has been presented suggesting that a red, hot, swollen site was caused by anything other than vaccination in this case. Further, Dr. Lancaster testified at the entitlement hearing that he would expect L.R. to experience pain from vaccination for one to two days. *Id.* at 191. So while it is possible that L.R.'s thigh pain constituted the onset of his anti-NMDAR encephalitis, I am not able to parse when pain from vaccination dissipated and pain from anti-NMDAR encephalitis began. At a minimum, the first two days of thigh pain that L.R. experienced can be reasonably attributed to vaccination, as conceded by Dr. Lancaster. Based on the above, there is not preponderant evidence that leg pain before three and one half days post-vaccination, constituted the onset of L.R.'s condition. I find there *is* preponderant evidence that L.R.'s altered mental state, late in the evening of August 7, 2014, represented the onset of his disease course.

2. Medically-Acceptable Timeframe

Dr. Steinman has opined that between three to four days is a medically appropriate

temporal interval between vaccination and onset of anti-NMDAR encephalitis. Second Steinman Rep. at 6; Tr. at 108-09. He primarily bases his opinion on language from the 2012 IOM.

[I]n a typical immune response to an antigen exposure, the latency between the first (primary) exposure and development of the primary response is characterized by a lag phase, logarithmic phase, and plateau phase. The lag phase is characterized by the initial activation of B and T cells upon encounter with the antigen for which they are specific, and this triggers the cells' differentiation into effector and memory cells. The lag phase between primary exposure to an antigen and the logarithmic phase is classically thought to be 4 to 7 days, but it varies depending on route of exposure and the antigen itself. For B cells, the logarithmic phase is characterized by an increase in serum antibody levels that classically is logarithmic. The plateau phase is characterized by the maintenance of peak antibody levels for a length of time that is followed by a decline in the serum antibody levels. For many antigens the latency (lag phase) between primary exposure and development of the primary antibody response is 7 to 10 days. Due to the development of memory B and T cells during the primary immune response, the latency between subsequent exposure to the antigen and development of the immune response will usually be shorter. The lag phase is generally 1 to 3 days; the logarithmic phase of the secondary antibody response occurs over the next 3 to 5 days. As mentioned for the primary immune response, these time periods will vary depending on the route of exposure, the timing of the subsequent exposure, the antigen itself, and the antigen dose. While this discussion is not specific to a particular antigen, it can be used as a reference point for the latency between antigen exposure and the initiation of some of the immune-mediated mechanisms described below.

IOM at 57-58. Dr. Steinman testified this means that the antibody response “really revs up” in days three to five. Tr. at 110. He opined that anti-NMDAR encephalitis could begin during this period of time. *Id.*

Dr. Lancaster disagreed. He testified that three to four days after vaccination was too soon for L.R. to experience signs and symptoms associated with anti-NMDAR encephalitis.

And yesterday we were discussing that a recall response might begin in four days, but that would mean that you're starting to detect a slight increase in antibody production against the target antigen within four days, not necessarily that you would have symptoms of an autoimmune neurologic disease within four days, which is a different issue.

Tr. at 185. According to Dr. Lancaster, before the disease process would begin, the patient would first need to experience proliferation of autoantibodies in the brain that built up to a level high enough level to cause symptoms. *Id.* He opined this would not occur within four days. *Id.*

Special masters have previously considered the appropriate length of time it would take for a recall response to cause disease onset. *Gardner v. Sec'y of Health & Hum. Servs.*, No. 17-1851V, 2023 WL 9288070, at *46 (Fed. Cl. Spec. Mstr. Dec. 21, 2023) (determining that several days was

an appropriate temporal interval for a post-vaccination recall response); *McGill v. Sec’y of Health & Hum. Servs.*, No. 15-1485V, 2023 WL 3813524, at *36, fn25 (Fed. Cl. Spec. Mstr. May 11, 2023) (concluding that a recall response would not explain onset of disease nine hours after vaccination, but noting that Respondent’s “explanation of “days to weeks” long latency” was not compelling); *Forrest v. Sec’y of Health & Hum. Servs.*, No. 14-1046V, 2019 WL 925495, at *8-9 (Fed. Cl. Spec. Mstr. Jan. 28, 2019) (finding that a recall response would not result in onset of transverse myelitis as soon as 36 hours after flu vaccination); *Quackenbush-Baker v. Sec’y of Health & Hum. Servs.*, No. 14-1000V, 2018 WL 1704523, at *20-23 (Fed. Cl. Spec. Mstr. Mar. 14, 2018) (concluding petitioner’s recall response to vaccination resulted in disease onset 40-41 hours later). I did not find a case that rejected the notion that a recall response could cause disease three to four days after vaccination.¹³

I find that Petitioner has presented preponderant evidence in support of her contention that a recall response could result in the onset of anti-NMDAR encephalitis within three to four days after vaccination. Dr. Steinman’s expert opinion supported by the IOM allow her to meet this burden. Petitioner has presented preponderant evidence in support of the third *Althen* prong.

VIII. Conclusion

Petitioner has my sympathy for L.R.’s ongoing medical condition and for her suffering during his hospitalization. However, after my careful evaluation of all the evidence submitted in this matter, including the medical records, the testimony, as well as the experts’ opinions and medical literature, I conclude that Petitioner has not shown by preponderant evidence that L.R.’s condition was caused by the vaccines he received. **The petition is therefore DISMISSED. The clerk shall enter judgment accordingly.**¹⁴

IT IS SO ORDERED.

s/ Katherine E. Oler

Katherine E. Oler
Special Master

¹³ This is consistent with the Vaccine Injury Table, which provides that development of GBS three to 42 days after the flu vaccine is a table injury. 42 C.F.R. § 100.3(a).

¹⁴ Pursuant to Vaccine Rule 11(a), the parties may expedite entry of judgment by each filing (either jointly or separately) a notice renouncing their right to seek review.